Curvilinear \( VO_2 \):Power Output Relationship in a Ramp Test in Professional Cyclists: Possible Association with Blood Hemoglobin Concentration

Alejandro Lucía*†, Jesús Hoyos†‡, Alfredo Santalla*, Margarita Pérez*†, and José L. Chicharro†§

* Departamento de Ciencias Morfológicas y Fisiología, Universidad Europea de Madrid, Spain; † Unidad de Investigación en Fisiología del Deporte, Universidad Complutense, Madrid, Spain; ‡ Asociación Deportiva Banesto, Madrid, Spain; and § Departamento de Enfermería, Universidad Complutense, Madrid, Spain

Abstract: The purpose of this study was to determine (1) if there exists an additional, nonlinear increase (\( \Delta VO_2 \)) in the oxygen uptake observed (\( VO_2_{obs} \)) at the maximal power output reached during a ramp cycle ergometer test and that expected (\( VO_2_{exp} \)) from the linear relationship between \( VO_2 \) and power output below the lactate threshold (LT) in professional riders, and (2) the relationship between \( \Delta VO_2 \) and possible explanatory mechanisms. Each of 12 professional cyclists (25±1 years; \( VO_2_{max} \): 71.3±1.2 ml · kg\(^{-1} \) · min\(^{-1} \)) performed a ramp test until exhaustion (power output increases of 25 W · min\(^{-1} \)) during which several gas-exchange and blood variables were measured (including lactate, HCO\(_3\)\(^{-} \) and K\(^{+} \)). \( VO_2 \) was linearly related to power output until the LT in all subjects. Afterward, a nonlinear deflection was observed in the \( VO_2 \):power output relationship (\( \Delta VO_2 = -492±55 \) ml · min\(^{-1} \) and \( p<0.05 \) for \( VO_2_{obs} \) vs. \( VO_2_{exp} \)). A significant negative correlation was encountered between \( \Delta VO_2 \) and resting hemoglobin levels before the tests (\( r=-0.61; \ p<0.05 \)). In conclusion, professional cyclists exhibit an attenuation of the \( VO_2 \) rise above the LT. [Japanese Journal of Physiology, 52, 95–103, 2002]

Key words: cycling, lactate, potassium, hemoglobin, \( P_{50} \), \( VO_2 \) kinetics.

Previous research with healthy, not highly trained humans has shown that the \( VO_2 \):workload relationship shows a nonlinear increase after the lactate threshold (LT) is exceeded during incremental exercise tests [1–7]. For instance, \( VO_2_{max} \) values obtained from gradual protocols can be significantly higher (9–17%) than the expected \( VO_2 \) at the final workload predicted from the previous linear relationship between \( VO_2 \) and workload below the LT [4, 6, 7].

Although some reports have studied the etiology of this nonlinear increase in the \( VO_2 \):power output relationship during incremental exercise (also termed “excess” \( VO_2 \)) [1–7], some controversy exists and more research is needed. Jones et al. [3] reported a causal relationship between lactic acidosis and the “excess” \( VO_2 \) during gradual running tests, but other reports using cycle ergometer tests have showed no link between both phenomena [4, 7]. On the other hand, the occurrence of the “excess” \( VO_2 \) remains to be confirmed in top-level endurance athletes.

The main purpose of this study was to determine if there exists a nonlinear increase in the \( VO_2 \):power output relationship of professional cyclists after the LT is exceeded during an incremental (ramp) protocol. The influence of some possible causal mechanisms of the \( VO_2 \) response above the LT was also analyzed.

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Correspondence should be addressed to: Alejandro Lucía, Departamento de Ciencias Morfológicas y Fisiología, Universidad Europea de Madrid, E-28670 Madrid, Spain. Fax: +34–916167568, E-mail: alejandro.lucia@mrfs.cisa.uem.es

**METHODS**

**Subjects.** After giving their written informed consent, 12 top-level professional road cyclists were selected as subjects for this investigation. Several of the subjects are among the best cyclists in the world (including a former world champion and winners of major professional tour races). Their mean (±SEM) age, height, and weight were: 25±1 years, 181.4±2.1 cm, and 70.4±1.5 kg, respectively.

**Exercise protocol.** Each subject performed an exercise test (ramp protocol) until exhaustion on a cycle ergometer (Ergometrics 900; Ergo-line; Barcelona, Spain) conducted in our laboratory during the months of December and January. All tests were performed under similar environmental conditions (20 to 24°C, 45 to 55% relative humidity). The subjects were cooled with a fan throughout the exercises. This type of protocol has been used for the physiological evaluation of professional cyclists in several previous studies [8–12]. After the cyclists had a 2 min rest sitting on the cycle ergometer, the test started at 25 W and the workload was increased by 25 W·min⁻¹. The subjects adopted the conventional sitting position during the duration of the tests. They were allowed to choose their preferred cadence within a range of 70–90 rpm. A recent report has shown that during actual racing, the preferred pedaling cadence of professional riders ranges between 70 rpm (hill climbs) and 90 rpm (flat terrains or individual time trials) [10]. The subjects used a pedal-frequency meter to maintain this range of cadences. Each test was terminated when pedal cadence could not be maintained at 70 rpm or more. Verbal encouragement was given to the subjects to continue the test until exhaustion. All subjects had had previous experience with this type of testing.

Several data obtained from these same tests and cyclists have been reported in a recent study in which we compared both the \( \dot{V}O_2 \) kinetics across all workloads (i.e., slope of the \( \dot{V}O_2 \):power output relationship) and the mechanical efficiency observed in the present subjects and a group of elite amateur cyclists [12].

**Measurements during the tests.** During the tests, heart rate (HR, in beats·min⁻¹) was continuously recorded and gas exchange data were continuously collected by using an automated breath-by-breath system (Mvmax 29C; Sensormedics; Yorba Linda, CA).

Capillary blood samples (50 μl) were taken from fingertips (sampling period of 15–20 s) at rest every 3 min throughout the test (at the end of 75 W, 150 W, 225 W, etc.), and immediately after the termination of exercise for the measurement of lactate concentration (BLa) with an automated analyzer (YSI 1500; Yellow Springs Instruments; Yellow Springs, O). The lactate threshold (LT) was determined by examining the BLa:power output (W) relationship during the tests according to the methodology described by Weltman and co-workers [13]. This method defines the power output corresponding to LT as the highest one not associated with a rise in BLa above baseline. This always occurred just before the curvilinear increase in BLa observed at subsequent exercise intensities. An increase of at least 0.5 mM BLa was required for the determination of the LT.

A 21-gauge butterfly needle was inserted into the antecubital vein of each subject before the tests. Blood samples were collected at rest, every 3 min throughout the tests (at the end of 75 W, 150 W, 225 W, etc.), and immediately after the termination of exercise. During each sampling period (~15 s), a 1-ml aliquot was initially withdrawn to clear the catheter and a 1.5 ml blood sample was subsequently collected with a heparinized syringe to measure the following variables with an automated blood gas analyzer (ABL725; Radiometer; Copenhagen, Denmark): hemoglobin concentration ([Hb]), hematocrit (Hct), fractional oxygen saturation of blood \( (P_{Vo_2}) \), partial pressure of oxygen \( (P_{Vo_2}) \), carbon dioxide \( (P_{vCO_2}) \), pH, and bicarbonate \( [HCO_3^-] \) and K⁺ concentrations. The \( P_{Vo_2} \) at which 50% of the hemoglobin is oxygenated was calculated both in vivo \( (P_{50, in vivo}) \) and under standard conditions \( (pH=7.4; \) temperature=37°C; and base excess=0) (standard \( P_{50} \)). Standard \( P_{50} \) was computed by using the equations previously reported by Severinghaus [14]. Based on ethical considerations, we considered the peripheral (antecubital) vein appropriate for the purpose of the study and avoided more invasive sampling, e.g., the use of the central venous or arterial catheters. Nevertheless, the use of samples from arm veins during cycle ergometry exercise must be kept in mind for a correct interpretation of our results. For instance, K⁺ concentration in the antecubital vein is the result of K⁺ coming to the arm from the body after the subtraction of K⁺ taken up in the arm and the addition of K⁺ released from the arm [15]. The latter is expected to be low in the cycle ergometer exercise, which mainly involves the lower limb muscles. Thus the measurement of K⁺ concentration in the arm veins should not overestimate the K⁺ concentration in the central venous vessels. It must also be noted that the K⁺ concentration in the arm veins is expected to be slightly delayed compared with changes occurring in the leg muscles and central vessels [15].

**Data analysis.** We used the same mathematical
approach as Zoladz et al. [7] did to describe the \( VO_2 \) response during the tests. The values of \( VO_2 \) (60-s average) between the 3rd min of exercise and the LT were fitted to a regression line. We then calculated the expected \( VO_2 \) (\( VO_{2\text{exp}} \), in \( \text{ml} \cdot \text{min}^{-1} \)) at the end of the test (average of the last 60 s) and \( VO_{2\text{exp}} \) was abbreviated as “\( \Delta VO_2 \)” [7].

To study the possible determinants of \( \Delta VO_2 \), we also calculated the magnitude of the differences between the values of HR, pulmonary ventilation (\( V_E \)), standard \( P_{50} \), [\( \text{HCO}_3^- \)], \( K^+ \) at the maximal power output, and those corresponding to the power output eliciting the LT, which we called \( \Delta HR \), \( \Delta V_E \), \( \Delta P_{50} \), \( \Delta [\text{HCO}_3^-] \), and \( \Delta K^+ \). The difference between \( \text{BLa} \) at the LT and that obtained at the end of exercise was measured and labeled \( \Delta \text{BLa} \).

A Student’s \( t \)-test for paired data was applied to determine the significance of the observed differences between \( VO_{2\text{obs}} \) and \( VO_{2\text{exp}} \). Pearson product–moment correlation coefficients were calculated to determine whether there was a significant relationship between \( \Delta V_O2 \), on the one hand and each of the following variables obtained from the exercise tests, i.e., \( \Delta HR \), \( \Delta V_E \),... and \( \Delta K^+ \) were also used to determine which of the aforementioned variables (\( \Delta HR \), \( \Delta V_E \),... \( \Delta \text{BLa} \); maximal values of HR, \( V_E \),... and \( \text{BLa} \); and rest values of \([\text{Hb}] \) and Hct) were predictor variables of \( \Delta VO_2 \).

The level of significance was set at 0.05 for all statistical analyses, and the results are expressed as means±SEM.

**RESULTS**

The individual and mean values of physiological variables recorded before the tests at the LT and maximal power output are shown in Tables 1–3, respectively. Individual and mean values of \( \Delta HR \), \( \Delta V_E \),... and \( \Delta K^+ \) are shown in Table 4. Figure 1 shows an example of the \( \Delta VO_2 \)-power output relationship and \( \Delta VO_2 \) and LT determination in one subject. The \( \Delta VO_2 \)-power output relationship of each subject as well as his \( \text{BLa} \) curve is shown in Fig. 2.

\( VO_2 \) was linearly related (\( p<0.05 \)) to power output until the LT in all subjects (Table 5). \( VO_{2\text{obs}} \) (5.021±105 ml·min\(^{-1}\)) was significantly lower (\( p<0.05 \)) than \( VO_{2\text{exp}} \) (5.513±110 ml·min\(^{-1}\)), with a negative mean value of \( \Delta VO_2 \) of \(-492±55 \text{ ml} \cdot \text{min}^{-1}\).

No significant correlation was found between \( \Delta VO_2 \) and each of the other variables obtained from the exercise tests, i.e., \( \Delta HR \), \( \Delta V_E \),... \( \text{BLa} \). In contrast, \( \Delta VO_2 \) was significantly correlated to resting \([\text{Hb}] \) (\( r=-0.61; p<0.05 \)) (Fig. 3). During the stepwise procedure, all variables except \([\text{Hb}] \) were removed from the model because they

Table 1. Individual and mean values of physiological variables before exercise.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Hct (%)</th>
<th>[Hb] (g·dl(^{-1}))</th>
<th>Standard ( P_{50} ) (mmHg)</th>
<th>( \text{BLa} ) (mM)</th>
<th>[( \text{HCO}_3^- )] (mM)</th>
<th>( K^+ ) (mM)</th>
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Mean (SEM) (0.8) (0.4) (0.3) (0.1) (0.4) (0.1)

\( \text{BLa} \) was measured in capillary blood. Standard \( P_{50} \), [\( \text{HCO}_3^- \)] and \( K^+ \) were measured in venous blood from the antecubital vein, not from the femoral vein. Hct, hematocrit; [Hb], hemoglobin concentration; standard \( P_{50} \), partial pressure of oxygen at which 50% of hemoglobin is oxygenated under standard conditions (\( \text{pH}=7.4; \text{temperature}=37^\circ \text{C}; \text{base excess}=0 \); \( \text{BLa} \), lactate concentration; [\( \text{HCO}_3^- \)], bicarbonate concentration; \( K^+ \), potassium concentration.
failed to meet the limits of the probability of $F$ to enter at $\leq 0.05$. The $R^2$ for the remaining variable, [Hb], was 0.37 (significant $F$ change $<0.05$), showing that 37% of the variance in $\Delta VO_2$ was explained by subjects’ [Hb]. These results also suggest that [Hb] was a predictor of $\Delta VO_2$.

**DISCUSSION**

In our subjects, the rate of $VO_2$ rise during gradual exercise (ramp protocol) decreased at moderate-to-high workloads, i.e., from the LT to the maximal attainable power output (from $\sim 300$ to $\sim 500$ W, respectively).

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**Table 2.** Individual and mean values of physiological variables at the lactate threshold.

<table>
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<tr>
<th>Subject</th>
<th>Power output (W)</th>
<th>HR (bpm)</th>
<th>$V_E$ (l·min$^{-1}$)</th>
<th>Hct (%)</th>
<th>[Hb] (g·dl$^{-1}$)</th>
<th>Standard $P_{50}$ (mmHg)</th>
<th>BLa (mm)</th>
<th>[HCO$_3^-$] (mM)</th>
<th>$K^+$ (mM)</th>
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<td>82.2</td>
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</table>

| Mean (SEM) | 312 (8) | 152 (3) | 87.2 (2.4) | 47.1 (0.7) | 15.4 (0.2) | 27.0 (0.1) | 1.6 (0.1) | 26.2 (0.4) | 4.5 (0.1) |

BLa was measured in capillary blood. Standard $P_{50}$, [HCO$_3^-$] and $K^+$ were measured in venous blood from the antecubital vein, not from the femoral vein. HR, heart rate; $V_E$, pulmonary ventilation; Hct, hematocrit; [Hb], hemoglobin concentration; standard $P_{50}$, partial pressure of oxygen at which 50% of hemoglobin is oxygenated under standard conditions (pH=7.4; temperature=37°C; base excess=0); BLa, lactate concentration; [HCO$_3^-$], bicarbonate concentration; $K^+$, potassium concentration.

**Table 3.** Individual and mean values of physiological variables at the maximal power output.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Power output (W)</th>
<th>$VO_2$ max (ml·kg$^{-1}$·min$^{-1}$)</th>
<th>HR (bpm)</th>
<th>$V_E$ (l·min$^{-1}$)</th>
<th>Hct (%)</th>
<th>[Hb] (g·dl$^{-1}$)</th>
<th>Standard $P_{50}$ (mmHg)</th>
<th>BLa (mm)</th>
<th>[HCO$_3^-$] (mM)</th>
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</table>

| Mean (SEM) | 508 (9) | 71.3 (1.2) | 196 (3) | 200.3 (5.4) | 49.2 (0.7) | 16.1 (0.2) | 29.4 (0.6) | 9.1 (0.3) | 17.4 (0.8) | 5.8 (0.1) |

BLa was measured in capillary blood. Standard $P_{50}$, [HCO$_3^-$] and $K^+$ were measured in venous blood from the antecubital vein, not from the femoral vein. HR, heart rate; $V_E$, pulmonary ventilation; Hct, hematocrit; [Hb], hemoglobin concentration; standard $P_{50}$, partial pressure of oxygen at which 50% of hemoglobin is oxygenated under standard conditions (pH=7.4; temperature=37°C; base excess=0); BLa, lactate concentration; [HCO$_3^-$], bicarbonate concentration; $K^+$, potassium concentration.
Fig. 1. Example of VO₂ response in one study subject with a considerable attenuation of the VO₂:power output relationship at high workloads (former world champion). LT, lactate threshold; BLa, blood lactate concentration in capillary blood; VO₂ exp, VO₂ expected at the maximal power output from the VO₂:power output linear relationship below the LT; VO₂ obs “true” VO₂ max at the maximal power output; ΔVO₂, difference between VO₂ obs and VO₂ exp.

Fig. 2. VO₂ and capillary blood lactate (BLa) responses in each of the 12 subjects. LT, lactate threshold. The dotted lines represent the mean power output (312±8 W) eliciting the LT.

Table 4. Individual and mean values of the differences between physiological variables at the maximal power output and at the lactate threshold.

<table>
<thead>
<tr>
<th>Subject</th>
<th>ΔHR (bpm)</th>
<th>ΔVE (l·min⁻¹)</th>
<th>ΔStandard P₅₀</th>
<th>ΔBLa (mM)</th>
<th>Δ[HCO₃⁻] (mM)</th>
<th>ΔK⁺ (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>36</td>
<td>138.1</td>
<td>3.1</td>
<td>8.5</td>
<td>-12.3</td>
<td>0.7</td>
</tr>
<tr>
<td>2</td>
<td>45</td>
<td>123.4</td>
<td>-0.7</td>
<td>5.4</td>
<td>-7.0</td>
<td>0.1</td>
</tr>
<tr>
<td>3</td>
<td>49</td>
<td>97.4</td>
<td>1.0</td>
<td>7.1</td>
<td>-7.9</td>
<td>1.7</td>
</tr>
<tr>
<td>4</td>
<td>31</td>
<td>86.6</td>
<td>6.0</td>
<td>7.3</td>
<td>-9.2</td>
<td>1.8</td>
</tr>
<tr>
<td>5</td>
<td>41</td>
<td>84.8</td>
<td>-0.9</td>
<td>8.4</td>
<td>-6.3</td>
<td>0.8</td>
</tr>
<tr>
<td>6</td>
<td>35</td>
<td>119.1</td>
<td>0.8</td>
<td>7.5</td>
<td>-6.4</td>
<td>0.6</td>
</tr>
<tr>
<td>7</td>
<td>47</td>
<td>138.6</td>
<td>3.4</td>
<td>8.1</td>
<td>-8.5</td>
<td>1.6</td>
</tr>
<tr>
<td>8</td>
<td>45</td>
<td>132.6</td>
<td>4.7</td>
<td>9.8</td>
<td>-10.1</td>
<td>2.0</td>
</tr>
<tr>
<td>9</td>
<td>52</td>
<td>100.4</td>
<td>4.1</td>
<td>5.0</td>
<td>-4.8</td>
<td>2.4</td>
</tr>
<tr>
<td>10</td>
<td>47</td>
<td>120.7</td>
<td>4.9</td>
<td>7.3</td>
<td>-11.6</td>
<td>1.4</td>
</tr>
<tr>
<td>11</td>
<td>52</td>
<td>122.4</td>
<td>0.0</td>
<td>7.7</td>
<td>-9.1</td>
<td>1.7</td>
</tr>
<tr>
<td>12</td>
<td>48</td>
<td>92.5</td>
<td>0.2</td>
<td>7.4</td>
<td>-12.6</td>
<td>1.2</td>
</tr>
</tbody>
</table>

| Mean (SEM) | 44 (5.7) | 113.1 (0.7) | 2.2 (0.4) | 7.5 (0.7) | -8.8 (0.7) | 1.3 (0.2) |

BLa was measured in capillary blood. Standard P₅₀, [HCO₃⁻] and K⁺ were measured in venous blood from the antecubital vein, not from the femoral vein. The symbol “Δ” represents the magnitude of the differences between the value of each variable (i.e., HR, VE,..., K⁺) at the maximal power output and that corresponding to the power output eliciting the lactate threshold (i.e., ΔHR, ΔVE,..., ΔK⁺). HR, heart rate; VE, pulmonary ventilation; Hct, hematocrit; [Hb], hemoglobin concentration; standard P₅₀, partial pressure of oxygen at which 50% of hemoglobin is oxygenated under standard conditions (pH=7.4; temperature=37°C; base excess=0); BLa, lactate concentration; [HCO₃⁻], bicarbonate concentration; K⁺, potassium concentration.
Another finding of this investigation was that the magnitude of this attenuation in the \( VO_2 \) response is partly associated with oxygen availability to working muscles, namely hemoglobin concentration. Other potential contributors to the \( VO_2 \) response above the LT in humans that are not highly trained (such as lactate and lactic acidosis, cardiorespiratory work, and \( K^+ \)) seemed to play no role here.

Scarce data from top-level endurance athletes are available in this area of research. The occurrence of a deflection in the \( VO_2 \) rise above the LT in our group of top-level cyclists (thus the lack of occurrence of the “excess” \( VO_2 \) phenomenon) differs from that previously described in research using gradual exercise and subjects with a lower fitness level [1–7]. One reason could apparently lie in the ramp protocol chosen for the present investigation (workload increases of 25 W·min\(^{-1}\)). The present protocol has been used in numerous reports with elite cyclists [8–12], but it differs from that used in most studies showing an “excess” \( VO_2 \) in not humans that are not well trained, i.e. workload increases every 3–4 min [1, 3, 6, 7]. Nevertheless, in a recent study from our laboratory in which we used a ramp protocol comparable to the present one (workload increases of 5 W/15 s, averaging 20 W·min\(^{-1}\)) and \( \Delta VO_2 \) was measured following the same methodology applied here, a significant “excess” \( VO_2 \) was found in sedentary young males [4]. In this previous report, \( VO_2 \) obs was 9% higher than \( VO_2 \) exp.

The somewhat unique \( VO_2 \) response of the subjects in the present study reflects one of the main adaptations to professional road cycling, that is, a great efficiency/economy, especially at high workloads, which partly accounts for their greater performance compared to amateur riders [9, 12]. Considering the high fitness level of the subjects’, (e.g., former world champion, some of the best climbing specialists in the world, etc.), we could draw an analogy to endurance running, a sport clearly dominated by Kenyan runners. It appears that their superior performance during the past decades compared to their European counterparts might be attributable, at least partly, to their greater running economy [16]. The high economy/efficiency of professional cyclists might contribute to their documented ability to sustain extremely high workloads over long periods, e.g., average power output >400 W during the 1 h record in a velodrome [17] or during

### Table 5. The linear relationship (\( p<0.05 \)) between \( VO_2 \) and power output (W) below the lactate threshold for each subject, where \( x=\text{power output (W)} \) and \( y=\text{VO}_2 \text{ (ml·min}^{-1}\text{)} \): Individual values of \( \text{VO}_2 \text{ obs, VO}_2 \text{ exp, and } \Delta \text{VO}_2 \) are also shown.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Regression</th>
<th>( r^2 )</th>
<th>( \text{VO}_2 \text{obs} ) (ml·min(^{-1}))</th>
<th>( \text{VO}_2 \text{exp} ) (ml·min(^{-1}))</th>
<th>( \Delta \text{VO}_2 ) (ml·min(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>( y=390.5+9.893x )</td>
<td>0.998</td>
<td>5,114</td>
<td>5,634</td>
<td>-520</td>
</tr>
<tr>
<td>2</td>
<td>( y=553.5+9.416x )</td>
<td>0.998</td>
<td>5,470</td>
<td>5,543</td>
<td>-73</td>
</tr>
<tr>
<td>3</td>
<td>( y=496.5+10.463x )</td>
<td>0.996</td>
<td>5,790</td>
<td>6,408</td>
<td>-618</td>
</tr>
<tr>
<td>4</td>
<td>( y=746.3+9.711x )</td>
<td>0.998</td>
<td>5,368</td>
<td>5,875</td>
<td>-507</td>
</tr>
<tr>
<td>5</td>
<td>( y=556.0+9.997x )</td>
<td>0.998</td>
<td>4,643</td>
<td>5,155</td>
<td>-512</td>
</tr>
<tr>
<td>6</td>
<td>( y=440.5+9.787x )</td>
<td>0.998</td>
<td>4,760</td>
<td>5,138</td>
<td>-378</td>
</tr>
<tr>
<td>7</td>
<td>( y=495.0+10.364x )</td>
<td>0.998</td>
<td>4,949</td>
<td>5,580</td>
<td>-631</td>
</tr>
<tr>
<td>8</td>
<td>( y=431.0+10.377x )</td>
<td>0.998</td>
<td>4,806</td>
<td>5,406</td>
<td>-600</td>
</tr>
<tr>
<td>9</td>
<td>( y=440.0+9.431x )</td>
<td>0.998</td>
<td>4,613</td>
<td>4,920</td>
<td>-307</td>
</tr>
<tr>
<td>10</td>
<td>( y=349.5+10.124x )</td>
<td>0.998</td>
<td>4,789</td>
<td>5,614</td>
<td>-825</td>
</tr>
<tr>
<td>11</td>
<td>( y=431.5+9.603x )</td>
<td>0.998</td>
<td>5,108</td>
<td>5,471</td>
<td>-363</td>
</tr>
<tr>
<td>12</td>
<td>( y=558.5+9.712x )</td>
<td>0.998</td>
<td>4,840</td>
<td>5,414</td>
<td>-574</td>
</tr>
</tbody>
</table>

Mean±SEM 5,021±105 5,513±110 492±55

\( VO_2 \) exp, \( VO_2 \) expected at the maximal power output from the \( VO_2 \) : power output linear relationship below the lactate threshold; \( VO_2 \) obs, “true” \( VO_2 \) max at the maximal power output; \( \Delta VO_2 \), difference between \( VO_2 \) obs and \( VO_2 \) exp.

**Fig. 3. Correlation between \( \Delta VO_2 \) and blood hemoglobin concentration ([Hb]).**
time trials [18]. These athletes, indeed, exhibit a considerable resistance to the fatigue of recruited motor units, at least at high submaximal intensities [9]. This adaptation is probably attained after years of highly demanding training and competition (i.e., ~35,000 km per year and ~90 competition days) as suggested in previous research that compared the physiological responses of professional cyclists during ramp exercise with those of their elite amateur counterparts [9, 12]. Power outputs above LT (~300 W), on the other hand, are similar to those expected during the most important phases of actual racing (i.e., time trials and high mountain passes) [18, 19], to which is oriented much cyclist training. The latter could partly explain the attenuation of the VO2:power output relationship above the LT compared to lower intensities. Similarly, highly trained runners show low economy when running at speeds below their race pace [20].

The “excess” nonlinear increase, which occurs in the VO2:power output relationship of most well-trained humans above the LT is mostly linked to the recruitment of Type II fibers, at least during constant-load tests [21]. Since our subjects did not show this response, one may speculate that they have a great proportion of Type I fibers in the main muscle involved in cycling, i.e., the vastus lateralis muscle. Previous research with competitive cyclists has indeed shown that cycling efficiency during heavy exercise (>LT) is positively related to the percentage of Type I fibers in the aforementioned muscle [22]. A high resistance to the fatigue of slow motor units could have also been involved, which would allow professional cyclists to reach moderate-to-high workloads (>LT) before a significant recruitment of less-efficient Type II fibers occurs. In fact, previous EMG research with elite cyclists [11] indirectly suggests that a significant recruitment of Type IIX fibers is not apparent in these athletes until near-maximal workloads (~90% VO2max).

Except for oxygen availability to working muscles (discussed below), no other physiological variables (e.g., cardiorespiratory work, potassium, lactate, and/or lactic acidosis) seemed to influence the VO2 response of our subjects at high workloads. Indeed, we found no correlation between ΔVO2 on the one hand and any of the variables related to cardiorespiratory work on the other (ΔVE and ΔHR). Previous research, however, suggests the involvement of lactate and/or lactic acidosis in the “excess” VO2, which occurs in not highly trained humans during incremental protocols [3]. For instance, the reduction in pH associated with lactic acidosis (as well as increased intramuscular temperature) causes a rightward shift in the oxyhemoglobin dissociation curve to the right because of the Bohr effect and may thus permit the continued VO2 increase [23]. In the present investigation, however, we found no correlation between ΔVO2 and several indicators of blood lactate accumulation and lactic acidosis, such as ABLa or BLa, at the end of exercise, nor did we find any correlation between ΔVO2 and Δ standard P50. Taken together, these results suggest that lactate and lactic acidosis per se do not increase whole-body VO2 in highly fit endurance athletes during gradual exercise. But it must also be kept in mind that the appearance or accumulation of lactate in the blood (which occurred in all subjects) does not necessarily imply the occurrence of type II fiber recruitment [24]. The recruitment of type II fibers, indeed, could be linked to the enhanced activity of other “anaerobic” energy pathways, i.e., the purine nucleotide cycle [24]. On the other hand, exercise-induced hyperpotassemia did not seem to influence VO2 kinetics. During intense exercise, muscle fibers release K+, and the plasma concentration of this ion increases, i.e., to 5.8±0.1 mM at the end of the tests. Although previous research with animals and humans has shown a positive correlation between blood K+ concentration and muscle metabolic rate [21], Poole et al. [25] found no parallelism between VO2 and K+ kinetics during constant-load, heavy exercise. To date little is known about the possible link between exercise-induced hyperpotassemia and the VO2:power output relationship at the high workloads of an incremental test, but our findings suggest no relationship between both variables. Concerning the latter, nevertheless, the fact that blood samples were collected from a peripheral (arm) vein must be kept in mind when interpreting our results, as mentioned above (see METHODS). During cycle ergometer tests, for example, the concentration of K+ in arm veins is expected to be slightly delayed compared with changes occurring in the leg muscles and central vessels [15].

Hemoglobin concentration, however, was negatively correlated to ΔVO2, suggesting, at least partly, an association between O2 delivery to working muscles and cycling efficiency in highly trained riders. In agreement with our findings, MacDonald and co-workers found that the magnitude of the “VO2 slow component” (i.e., the increase in VO2 from min 3 to min 6) of healthy humans during heavy constant-load cycle-ergometer exercise (above the ventilatory threshold or VT) was significantly lower when oxygen transport was artificially increased with hyperoxic breathing (inspiratory oxygen fraction of 0.70), compared to normoxic conditions [26]. Taken together, their findings and the present ones suggest that improved oxy-
blood O₂ carrying capacity could also have a positive
mance, our findings suggest that increases in the
crease in the
metabolism.
cardiac output places a limiting ceiling on peak mus-
cle mass (i.e., cycle ergometry), especially in subjects
as highly trained as the present ones are [28]. Thus in-
creases in blood O₂ transport capacity are expected to
improve maximal performance in top-level athletes
[19]. Besides this potential effect on maximal perfor-
manship at high workloads, it is not possible to estimate at
workload cardiac output reached a ceiling and
output relationship at high workloads (VT).

The rate of VO₂ rise during a ramp protocol in professional cyclists decreases after the LT has been reached. This response is different from the one exhibited by most individuals (i.e., a nonlinear increase in the VO₂:power output relationship above the LT). Although further research is needed, increases or decreases in oxygen-carrying capacity could have a positive or detrimental effect, respectively, on cycling efficiency at the aforementioned workloads.

We thank José Ramón Echevarría for his great technical as-
sistance during the tests.

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gen transport capacity, whether artificially increased
(e.g., through hyperoxic breathing) or because of in-
creased hemoglobin concentration within physiological
limits, reduces the increase rate of the VO₂:power
capacity in working muscles [27]. It is commonly accepted that maximal
cardiac output places a limiting ceiling on peak mus-
cle blood flow and O₂ delivery to working muscles
during incremental exercise tests involving large mus-
cle mass (i.e., cycle ergometry), especially in subjects
as highly trained as the present ones are [28]. Thus in-
creases in blood O₂ transport capacity are expected to
improve maximal performance in top-level athletes
[19]. Besides this potential effect on maximal perfor-
mance, our findings suggest that increases in the
blood O₂ carrying capacity could also have a positive
effect on submaximal performance at moderate-to-
high workloads. Since we did not measure cardiac
output in our subjects, it is not possible to estimate at
which workload cardiac output reached a ceiling and
thus could influence VO₂ kinetics, i.e., inducing a re-
cruitment of Type II fibers that can rely on anaerobic
metabolism.

In conclusion, the rate of VO₂ rise during a ramp
protocol in professional cyclists decreases after the LT
has been reached. This response is different from the
one exhibited by most individuals (i.e., a nonlinear in-
crease in the VO₂:power output relationship above the
LT). Although further research is needed, increases or
decreases in oxygen-carrying capacity could have a
positive or detrimental effect, respectively, on cycling
efficiency at the aforementioned workloads.

We thank José Ramón Echevarría for his great technical as-
sistance during the tests.

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