Effects of concurrent endurance and strength training on running economy and \( \dot{V}O_2 \) kinetics

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ABSTRACT

MILLET, G. P., B. JAOUEN, F. BORRANI, and R. CANDAU. Effects of concurrent endurance and strength training on running economy and \( \dot{V}O_2 \) kinetics. *Med. Sci. Sports Exerc.*, Vol. 34, No. 8, pp. 1351–1359, 2002. Purpose: It has been suggested that endurance training influences the running economy (CR) and the oxygen uptake (\( \dot{V}O_2 \)) kinetics in heavy exercise by accelerating the primary phase and attenuating the \( \dot{V}O_2 \) slow component. However, the effects of heavy weight training (HWT) in combination with endurance training remain unclear. The purpose of this study was to examine the influence of a concurrent HWT+endurance training on CR and the \( \dot{V}O_2 \) kinetics in endurance athletes. Methods: Fifteen triathletes were assigned to endurance+strength (ES) or endurance-only (E) training for 14 wk. The training program was similar, except ES performed two HWT sessions a week. Before and after the training period, the subjects performed 1) an incremental field running test for determination of \( \dot{V}O_2\text{max} \) and the velocity associated (\( \dot{V}O_2\text{max} \)), the second ventilatory threshold (VT2); 2) a 3000-m run at constant velocity, calculated to require 25% of the difference between \( \dot{V}O_2\text{max} \) and VT2, to determine CR and the characteristics of the \( \dot{V}O_2 \) kinetics; 3) maximal hopping tests to determine maximal mechanical power and lower-limb stiffness; 4) maximal concentric lower-limb strength measurements. Results: After the training period, maximal strength were increased \((P < 0.01)\) in ES but remained unchanged in E. Hopping power decreased in E \((P < 0.05)\). After training, economy \((P < 0.05)\) and hopping power \((P < 0.001)\) were greater in ES than in E. \( \dot{V}O_2\text{max} \text{ leg hopping stiffness and the } \dot{V}O_2 \text{ kinetics were not significantly affected by training either in ES or E. Conclusion: Additional HWT led to improved maximal strength and running economy with no significant effects on the \( \dot{V}O_2 \) kinetics pattern in heavy exercise. Key Words: ENERGY COST, MAXIMAL OXYGEN CONSUMPTION, OXYGEN UPTAKE SLOW COMPONENT, HOPPING POWER

The combined effects of concurrent strength and endurance training on the endurance performance of untrained (12,16,18,19,25) or trained athletes (13,14,22,23) have been extensively studied. There is evidence to suggest that endurance training inhibits maximal strength development, mainly a few weeks after commencement of a concurrent training regime (25). It has been suggested that although strength training does not interfere with the development of the maximal oxygen uptake (\( \dot{V}O_{2\text{max}} \)) (11–12,18), it could lead to improvement of endurance performance in untrained (11,12,18,19,25) or moderately trained athletes (13).

It has been well documented that the speed achieved in endurance competition relies not only on the rate of energy expenditure but also on the energy cost (CR) of the considered locomotion (9). It also appears that CR is a better predictor of endurance performance than \( \dot{V}O_{2\text{max}} \) in a homogeneous group of athletes (8). However, the effects of concurrent strength and endurance training on economy in well-trained endurance athletes are still unclear. It has been argued that strength training improves (13,14,23) or has no influence (29) on the economy. Paavolainen et al. (22,23) investigated the effects of explosive-strength training in well-trained athletes, but the effects of concurrent heavy weight training (HWT) and endurance training on economy have not been studied in elite athletes.

At the highest submaximal intensities, above the lactate threshold, CR represents only the aerobic contribution to the total energy expenditure (9), and this aerobic part tends to rise slowly. The slow component of oxygen uptake kinetics in long-term constant-rate exercise can be described as an increase in the energy expenditure above that predicted from submaximal \( \dot{V}O_2 \text{-work rate relationship, leading to a reduced work efficiency (10). Several studies (5,6,10,21,24,28) have shown that endurance training results in a change in \( \dot{V}O_2 \) kinetics, so that a shorter constant time of the primary phase (21,24) and a reduced amplitude of the \( \dot{V}O_2 \) slow component occurs (5,10,28). It was proposed that around 85% of the \( \dot{V}O_2 \) slow component is located at the muscular level (10) and that central factors have only a minor influence on its amplitude. A limited \( \dot{V}O_2 \) slow component has been suggested to be an important parameter of the endurance performance (10). However, to the best of our knowledge, very little is known on the effects of strength training on the characteristics of the \( \dot{V}O_2 \) kinetics, especially the parameters that described the primary phase and the slow component (29). Therefore, the purpose of
the present study was to examine the effects of a regime of maximal strength training, in combination with an existing endurance-training program, on the running economy in well-trained triathletes. Furthermore, this study was conducted to determine whether strength training influences the VO₂ kinetics during heavy constant-rate running exercise.

METHODS

Approach to the problem and experimental design. The hypothesis that combined HWT and endurance training would lead to greater lower limb strength, power, and stiffness and be more transferable to a better running economy than endurance-only training was tested. To answer this question and compare the impacts of a combined versus an endurance-only training, we chose two different training regimes, suitable for inclusion in the winter schedule of national and international-level triathletes, over a long period of 14 wk, a duration classically observed in the endurance sports. A field-based approach was applied to evaluate both realistic central and peripheral adaptations that could influence the performance.

Subjects. Fifteen well-trained subjects were randomly assigned to the endurance-strength training group (ES; N = 7) or to the endurance-only training group (E; N = 8). Seven of them, practicing at an international level (elite national team), were matched in the two groups (three in ES and four in E). All the subjects agreed to participate in the study on a voluntary basis. The study was approved by the institutional ethics committee, and all subjects provided written, voluntary, informed consent before participation. The subjects were all fully familiar with testing procedures, having regularly being tested as part of their training evaluation. The physical characteristics of the two groups are shown in Table 1.

Methods. Before and after a controlled training period, all subjects performed field and laboratory-based running and muscle function tests. The first test involved an incremental running test to exhaustion to determine the maximal oxygen uptake (VO₂max), the velocity associated with VO₂max (V VO₂max), the velocity associated to the second ventilatory threshold (V VO₂2), and the velocity associated to the intensity termed Δ25% (V Δ25%), corresponding to VO₂ V O₂2 plus 25% of the difference between VO₂ V O₂2 and VO₂max. The second test comprised a 3000-m run at a controlled constant V Δ25% preceded by 6 min at 75% V VO₂max to determine running energy cost at the two intensities (CR 75% and CR Δ25%) and record breath-by-breath VO₂ data to model VO₂ kinetics during exercise. A third series of tests included maximal hopping tests at a 2-Hz frequency to determine the maximal mechanical hopping power and lower-limb hopping stiffness. A forth series was maximal concentric lower-limb strength measurements.

General training. The training period lasted for 14 wk and was carried out during the winter period, when the subjects were not involved in any competitions. The training period started after a 10-wk preconditioning-orientation phase, where the subjects restarted consistent training. All athletes were experienced and members of a residential training center where they were under the control of professional trainers. Moreover, they recorded training exercises in a diary that was reviewed regularly. The vast majority of the training during this basic period was strictly aerobic, realized under 70% of VO₂ (Table 1). Stretching remained constant (1.6 ± 0.5 h·wk⁻¹) in the two groups.

Strength training. In addition to the endurance training, the ES group performed an HWT session of lower-limb muscles twice a week. Exercises (i.e., hamstring curl, leg press, seated press, parallel squat, leg extension, and heel raise) were exclusively focused on quadriceps, hamstrings, and calf muscles. Workouts consisted of two warm-up sets followed by three to five sets to failure of 3–5 reps. The training program was periodized and was composed of several 3-wk periods. In each of these periods, the number of sets increased (i.e., three in the first week, four in the second week, and five in the third week). The loads were calculated ≥ 90% one-repetition-maximal (1RM) and were progressively increased to maintain this range of repetitions per set. Reassessment of 1RM was completed by the ES group every 3 wk to maintain maximal loads over the whole training period.

Testing. The ES and E groups were examined before training and after the training period. The testing protocol was conducted over two consecutive days on a 400-m synthetic track, then an additional day in the following week in the laboratory for concentric strength and hopping tests. ES performed supplementary maximal strength tests at week 6.

Maximal concentric lower-limb strength measurements. Maximal concentric strength evaluation was performed using two exercises (half-squat and heel raise) by all athletes before and after the training period. After a warm-up workout, the subject’s near-maximal load was approximated by the trainer to be around 90% of the previous best load of the subject. The load was gradually increased until the subject could lift the resistance once but not twice. This load was therefore defined as 1RM. For the half-squat, the starting position was at a knee angle of 120°.
and the exercise was performed in a guided strength rack, ensuring maximal security. The amplitude of the movement was controlled by the trainer. For the heel-raise exercise, the starting position was a standing position with straight legs. Because the resistance was maximal, two assistants helped the subjects to position the bar correctly over the shoulders, to prevent any accident.

**Hopping tests.** Maximal vertical rebounds on both legs were executed by the subjects from a standing position at 2 Hz for 10 s before and after the training period. Subjects were instructed to rebound to the highest possible point with the smallest ground contact times and to keep hands on the hips throughout the hops (4,7). Flight time ($t_f$) and ground contact times ($t_c$) were recorded by an apparatus consisting of a digital timer connected to a contact mat (Powertimer, Newtest, Oulu, Finland) with an accuracy of 1/100 s. As described previously (4,7), the displacement of the center of mass during the flight ($h_f$) and the maximal mechanical power of the positive work (P) of the subjects were calculated:

$$h_f = (g \cdot t_f^2) / 8 \text{ (hop height, in m)}$$  

$$P = (m \cdot g \cdot t_f^2) / (4 \cdot t_c) \text{ (maximal hopping power, in W)}$$  

where $m$ the body mass of the subject, $t_f$ is the total time of the hop ($t_c = t_f + t_i$), and $g$ is the gravitational acceleration. The vertical stiffness of the lower limbs ($K_{vm}$; N·m·kg$^{-1}$) is the force change/length change ratio and was calculated as described previously (7):

$$K_{vm} = m \cdot g \cdot \omega \theta \text{ (lower limbs hopping stiffness, in kN·m·kg}^{-1}$$  

with $\tan(\pi - (\omega \theta / 2)) = t_i \cdot t_c$  

where $\omega \theta$ is the forced oscillation of the body while vertical hopping. With $t_i$ and $t_c$ as known variables, equation 4 can be solved and $\omega \theta$ determined.

**Track running tests.** The following respiratory gas-exchange variables were collected, using a breath-by-breath portable gas analyzer (Cosmed K4b$^2$, Rome, Italy): $\dot{V}O_2$, $\dot{V}CO_2$, pulmonary ventilation (VE), ventilatory equivalents for oxygen (VE/$\dot{V}O_2$) and carbon dioxide (VE/$\dot{V}CO_2$), end-tidal PO$_2$ (PET$\dot{O}_2$), and PCO$_2$ (PET$\dot{CO}_2$). Calibration procedures were performed before each test according to the manufacturer’s instructions. Heart rate (HR) was recorded by the K4b$^2$ via a portable HR monitor belt (Polar$^®$ Electro, Kempele, Finland). At the end of the tests, subjects indicated their rating of perceived exertion (RPE) using a 6–20 scale. All tests were preceded by a 5-min standing rest to determine the $\dot{V}O_2$ baseline ($\dot{V}O_2$). 

**Incremental test to exhaustion.** The subjects performed first the incremental test to exhaustion on a 400-m running track to determine the maximal oxygen uptake ($\dot{V}O_2$max), the velocity associated with $\dot{V}O_2$ ($\dot{V}O_2$max), and the velocity associated with the second ventilatory threshold ($\dot{V}O_2$VT2). In addition, the velocity ($\dot{V}O_2$) corresponding to $\dot{V}O_2$VT2 plus 25% of the difference between $\dot{V}O_2$VT2 and $\dot{V}O_2$max was calculated.

$$\Delta 25% = \dot{V}O_2VT2 + 0.25 \times (\dot{V}O_2max - \dot{V}O_2VT2).$$  

The initial velocity was set as 8 km·h$^{-1}$ under the estimated $\dot{V}O_2$max. The duration of the test was expected to be between 15 and 20 min. The increments of velocity were set at 0.5 km·h$^{-1}$ for stages of 1 min. The subject adjusted his velocity to sound signals and visual marks at each 20-m interval around the track. All subjects were familiarized with this procedure, having completed similar paced exercise sessions during training. All subjects were encouraged to perform their best effort. Breath-by-breath data were averaged over 30 s, and $\dot{V}O_2$max was defined as the highest 30-s value reached. $\dot{V}O_2$max was determined as the minimal velocity at which $\dot{V}O_2$max was reached. The second ventilatory threshold ($\dot{V}O_2$VT2) was defined by 1) a systematic increase in VE/$\dot{V}O_2$; 2) a concomitant nonlinear increase in the VE/$\dot{V}CO_2$, and 3) a decrease in the $\Delta$PET$\dot{O}_2$ (difference in the inspired and end-tidal O$_2$ pressure). $\dot{V}O_2$VT2 was determined by two independent observers.

**3000-m test at $\dot{V}O_2$max.** On the second day, the subjects performed a 3000-m run at $\dot{V}O_2$max. The subjects warmed up 6 min at ~60% $\dot{V}O_2$max followed by 6 min at a controlled $\dot{V}O_2$75% velocity, where $\dot{V}O_2$75% = 0.75 × $\dot{V}O_2$max is the intensity where a $\dot{V}O_2$ slow component has been previously observed (6). Before the start of the 3000 m, the subjects rested for 5 min to determine $\dot{V}O_2$, $\dot{CR}$75%, and $\dot{CR}$25% (in mL·O$_2$·kg$^{-1}$·min$^{-1}$) were calculated from the averaged 3rd- to 4th-min $\dot{V}O_2$ above basal metabolic rate (BMR), at respectively the $\dot{V}O_2$75% warm-up and the $\dot{V}O_2$25% 3000 m, as follows:

$$CR = (\dot{V}O_2 - 0.083) \times V^{-1}$$  

where $\dot{V}O_2$ is expressed in mL·kg$^{-1}$·s$^{-1}$, 0.083 mL·kg$^{-1}$·s$^{-1}$ is the y-intercept of the $\dot{V}O_2$ velocity relationship of young adults, and $V$ is expressed in m·s$^{-1}$. At posttraining, $\dot{V}O_2$25% was rationalized per minute.

**Kinematic variables and running leg stiffness.** Average stride frequency (SF, in Hz) and stride length (SL, in m) were recorded eight times, over a 100 m of each lap (SF × SL = average velocity over 100 m). The average values over the 3000-m were retained. The average posttraining running leg stiffness was approximated with the exponential model was used (1,3).

$$\dot{V}O_2 VT2 = A_1 \cdot (1 - e^{-t_2 \cdot td_2}) \cdot U_1 + A_2 \cdot (1 - e^{-t_1 \cdot td_1}) \cdot U_2$$  

Phase 2 (primary component) Phase 3 (slow component)  

where

$$U_1 = 0 \text{ for } t < td_1 \text{ and } U_1 = 1 \text{ for } t \geq td_1$$  

$$U_2 = 0 \text{ for } t < td_2 \text{ and } U_2 = 1 \text{ for } t \geq td_2$$  

$\dot{V}O_2$ is the $\dot{V}O_2$ at rest, $A_1$ and $A_2$ are the asymptotic amplitude, $td_1$ and $td_2$ are the time delays after the start of the exercise, $t_1$ and $t_2$ are the constant times, respectively, for the second and third phase. Because the primary phase is not distorted by any cardiodynamic influence, the first
residual errors between the modeled and the measured \( \dot{V}_O_2\) kinetics. The distribution of the residual sum of the mean squares of the differences between the modeled \( \dot{V}_O_2\) and the measured \( \dot{V}_O_2\) (B).

20 s were not taken account in the calculation of the parameters of the primary phase (\( A_1 \) and \( A_2 \)). The amplitude of the \( \dot{V}_O_2 \) slow component was defined as \( A_2 \).

\[
A_2' = A_2[1 - e^{-(t - t_0)/\tau_2}]
\]

where \( t_e \) is the time at the end of the exercise.

As described previously (3), the parameters of the model were calculated by an iterative procedure by minimizing the sum of the mean squares of the differences between the modeled \( \dot{V}_O_2\) and the measured \( \dot{V}_O_2\) (see subject 1 in Fig. 1A). The values of the measured breath-by-breath \( \dot{V}_O_2\) that were outside a three standard deviations range from the modeled \( \dot{V}_O_2\) were removed, representing less than 0.5% of the data collected. The time delay for the slow component phase (\( t_d_2 \)) was fixed to be higher than the time of the first exponential component for reaching \( A_1' \), where \( A_1' \geq 99% A_1 \). A Fisher test was used to determine the degree of significance of the exponential model. The distribution of residual errors between the modeled and the measured \( \dot{V}_O_2\) as a function of time was tested using linear and nonlinear regressions.

Statistical analysis. Paired \( t \)-tests were used to determine the significance of differences in the measured variables before versus after training. When the normality test failed, a Mann-Whitney rank sum test was performed between pre- and post-training variables. A repeated measures ANOVA was used to identify differences between the two groups of subjects, by examination of the group \( \times \) time interaction. Statistical power was determined to be from 0.57 to 0.69 for the sample sizes used at the 0.05 alpha level (SigmaStat, Jandel Corporation, San Rafael, CA). Effect size (ES) was calculated for each test and displayed for every significant effect. Pearson correlation coefficients were used to examine the relationships between change of economy and change of power, stiffness, or strength variables. The results are presented as means \( \pm \) SD. For all statistical analyses, a \( P \)-value of 0.05 was accepted as the level of statistical significance.

RESULTS

The main characteristics of the endurance training of the triathletes are presented in Table 1. No differences were observed in the training parameters between ES and E during the period studied.

The effect of training on the physiological variables during the incremental track-running test are shown in Table 2. No significant changes during the training period were ob-

<table>
<thead>
<tr>
<th>Incremental Test to Exhaustion</th>
<th>( \dot{V}<em>O_2</em>{peak} ) (mL·min(^{-1})·kg(^{-1}))</th>
<th>( V_T_2 ) (( % \dot{V}<em>O_2</em>{max} ))</th>
<th>HR (bpm)</th>
<th>RPE (points)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ES (( N = 7 ))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretraining</td>
<td>69.7 ± 3.6</td>
<td>88.4 ± 2.8</td>
<td>189 ± 10</td>
<td>16.0 ± 1.4</td>
</tr>
<tr>
<td>Posttraining</td>
<td>67.2 ± 4.4</td>
<td>89.1 ± 5.0</td>
<td>189 ± 11</td>
<td>18.3 ± 0.5</td>
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<tr>
<td>E (( N = 8 ))</td>
<td></td>
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</tr>
<tr>
<td>Pretraining</td>
<td>67.6 ± 6.4</td>
<td>89.3 ± 8.1</td>
<td>190 ± 5</td>
<td>16.5 ± 1.7</td>
</tr>
<tr>
<td>Posttraining</td>
<td>67.3 ± 5.8</td>
<td>88.3 ± 6.4</td>
<td>189 ± 5</td>
<td>16.5 ± 1.4</td>
</tr>
</tbody>
</table>

Values are means \( \pm \) SD. \( \dot{V}_O_2_{max} \) maximal oxygen uptake; \( V_T_2 \) second ventilatory threshold; HR, maximal heart rate; RPE, rating of perceived exertion.
TABLE 3. Measured and calculated parameters during the constant-velocity 3000-m test, before and after training in the endurance-strength (ES) and endurance-only (E) triathletes.

<table>
<thead>
<tr>
<th>3000-m at Constant Velocity</th>
<th>V_{O2max} (km·h^{-1})</th>
<th>HR (bpm)</th>
<th>RPE (points)</th>
<th>BL (mL·min^{-1}·kg^{-1})</th>
<th>td_{1} (s)</th>
<th>t_{1} (s)</th>
<th>A_{1} (mL·min^{-1}·kg^{-1})</th>
<th>td_{2} (s)</th>
<th>t_{2} (s)</th>
<th>A_{2} (mL·min^{-1}·kg^{-1})</th>
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<tbody>
<tr>
<td>ES (N = 7)</td>
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<tr>
<td>Pretraining</td>
<td>17.4 ± 0.9</td>
<td>181 ± 11</td>
<td>14.7 ± 1.1</td>
<td>11.2 ± 5.8</td>
<td>8 ± 7</td>
<td>13 ± 6*</td>
<td>46.4 ± 9.4</td>
<td>65 ± 39</td>
<td>61 ± 32*</td>
<td>6.7 ± 4.3</td>
</tr>
<tr>
<td>Posttraining</td>
<td>17.6 ± 0.8</td>
<td>185 ± 11</td>
<td>14.9 ± 0.9</td>
<td>12.0 ± 5.5</td>
<td>6 ± 7</td>
<td>15 ± 6</td>
<td>44.2 ± 9.9</td>
<td>83 ± 43</td>
<td>84 ± 76</td>
<td>5.1 ± 3.7</td>
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<tr>
<td>E (N = 8)</td>
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</tr>
<tr>
<td>Pretraining</td>
<td>17.2 ± 1.1</td>
<td>186 ± 5</td>
<td>14.8 ± 1.4</td>
<td>11.5 ± 4.1</td>
<td>8 ± 7</td>
<td>21 ± 6</td>
<td>46.1 ± 8.5</td>
<td>84 ± 83</td>
<td>91 ± 142</td>
<td>3.7 ± 3.3</td>
</tr>
<tr>
<td>Posttraining</td>
<td>17.5 ± 1.1</td>
<td>187 ± 5</td>
<td>15.1 ± 1.0</td>
<td>12.5 ± 4.6</td>
<td>7 ± 7</td>
<td>17 ± 9</td>
<td>47.9 ± 8.7</td>
<td>102 ± 83</td>
<td>165 ± 160</td>
<td>4.6 ± 3.7</td>
</tr>
</tbody>
</table>

Values are means ± SD. V_{O2max}, velocity associated with Δ25% = VT_{4} + 0.25 × (V_{O2max} − VT_{2}); HR, end-exercise heart rate; RPE, rating of perceived exertion; BL, baseline; td_{1} and td_{2}, time delays; t_{1} and t_{2}, time constants; A_{1} and A_{2}, amplitude of, respectively, the fast primary component and the slow component of the VO_{2} response. *P < 0.05 for differences between groups.
disregard
the training period suggests that the E group’s decrease of muscular power may have affected its running economy.

However, no direct measures of power during the $V_{\Delta 25\%}$ 3000 m were recorded. These results suggest that a large volume of endurance training can lead to a decrease of the neuromuscular characteristics, becoming therefore limiting factors of endurance efficiency, even in endurance athletes.

Alteration in the leg stiffness regulation and in the storage-recoil of energy with fatigue has been proposed as a cause of the decrease in the efficiency and in the economy of runners (15). A negative correlation between the stiffness of the dominant leg and CR has been reported (7). In the present study, no changes in contact time or vertical stiffness while hopping were observed. The type of strength performed during the training period (concentric contraction; >90% 1RM) is associated with lower mean power frequency, shift of the EMG, or rate of neural input than explosive-strength training (concentric and plyometric contractions; 40–50% 1RM), mainly caused by a slower movement velocity (17,23). Therefore, the characteristics of the strength training performed in the present study may not have been optimal for improving the stiffness in a maximal stretch-shortening cycle exercise like hopping, even if it caused a significant increase in concentric strength and power. Nevertheless, estimated running leg stiffness was higher after training in ES than in E, meaning that some adaptations could have occurred.

**Change of maximal strength.** The gains in maximal strength of the ES group (25% in the half-squats and 17% in the calf raise) were similar to the improvement showed in previous studies [i.e., 14.5% in ski ergometer test (13), 19.5% in leg press (16), 27% in parallel squats (11), and 33.8% in lower-body lifts (14)], whereas the E group exhibited no strength increase, as a result of the lack of strength training and the fact that they performed low-intensity (i.e., a low percentage of the maximal voluntary force) endurance training during the period studied. It is of interest to observe that the body weight did not change in either the ES or the E group, suggesting that little (if not at all) hypertrophy was caused by the strength training in ES. HWT contributes to strength gains mainly by two type of factors, primarily neural factors (increased activation, more efficient recruitment, motor unit synchronization, and excitability of the α-motor neurons or decreased Golgi tendon organ inhibition) in the early phase and hypertrophy on a longer term. However, after 2–6 wk, hypertrophy of Type IIa and IIb but also Type I muscle fibers were observed (26). Moreover, fast-twitch fibers type conversion from IIb to IIA was reported to occur as an effect of combined training (16,26). The decrease in the percentage of the Type IIB and the concomitant increase of the Type IIA fibers may lead to an increase of the oxidative capacity of the trained muscles. Moreover, Sale et al. (25) showed that the increase of the activity of the main oxidative enzymes (i.e., citrate synthase) and of the percentage of slow-twitch fibers in the muscles trained was similar in endurance-only and in combined endurance-strength groups. These results suggest that, even over a long period, the oxidative capacity of the lower-limb muscle are not affected by the inclusion of strength training into endurance training, supporting the concept of a possible “additive effect” of strength and endurance training. Combined training could also cause a “compromising effect” at the cellular level for both endurance and strength capabilities attenuating the adaptations (16). For example, Kraemer et al. (16) reported no changes in Type I and Iic fibers after a 12-wk combined training program, which may be related to an overtraining status as shown by the undesirable increase in cortisol.

**Change in $V_{VO2max}$** The ES group had a 2.7% improvement in $V_{VO2max}$ whereas the 2.2% change was not significant in the E group. However, because the interaction group × time was not significant, this difference cannot be attributed to the training difference between the two groups. For a statistical power of 0.8 and a difference in $V_{VO2max}$ of 0.49 ± 0.66 km·h⁻¹, the size of the group E should have been of 17 subjects for reaching the significance level of $α = 0.05$. Moreover, in the present study, the correlation between change in $V_{VO2max}$ and change in CR with the two groups pooled was weak ($r = -0.46; P = 0.09$), indicating that changes in CR would not explain to a great extent change in running speed at $V_{VO2max}$.

**Unchanged $VO2$ kinetics.** Most of the previous studies investigating the effects of training on the $VO2$ kinetics used a fixed time interval (6,28,29) and not a mathematical model (5). Bearden and Moffatt (2) showed that the use of a fixed interval led to an underestimation of the slow component. In the present study, a fixed 3– to 10-min interval would have resulted in a significantly different amplitude of the slow component than with the two-exponential component model. With the fixed interval, the amplitude was significantly ($P < 0.05$) underestimated (1.5 ± 1.5 vs 6.7 ± 4.3, 1.6 ± 2.6 vs 5.1 ± 3.7, 1.4 ± 3.0 vs 3.7 ± 3.3, 1.2 ± 1.7 vs 4.6 ± 3.7, respectively, for ES and E at pre- and post-training) when compared with the two-exponential model finally chosen.

Although the obvious interest to better understand whether additional strength training into an endurance training regime would cause different changes in the $VO2$ kinetics, to the best of our knowledge, only one study (29) has investigated the effect of strength training on the $VO2$ kinetics. In the present study, 14 wk of training did not change the $VO2$ kinetics neither in the ES nor in the E group. The results of the present study, showing no changes during the training period in either the E or the ES group in $t_d$, $\tau_1$, $A_1$, $t_d$, $\tau_2$, or $A_2$, are in line with the study of Womack et al. (29). One could assume that the muscular adaptations induced by the additional HWT were not pronounced enough to cause changes in the $VO2$ kinetics when compared with endurance training. However, in the two groups pooled, training did cause an improvement in the velocities ($P < 0.01$) without any change in the associated $V_{VO2max}$ or $V_{I2}$, meaning that most of the induced adaptations were located at the muscular level.

Acceleration of the $VO2$ adjustments at the onset of heavy exercise after endurance training was commonly reported in previous studies (21,24) but not in all (5). The present study
did not confirm that in already well-trained athletes training induces faster constant time of the primary phase. Carter et al. (5) reported no effects of training on phase 2 during heavy exercise in healthy subjects (pretraining $\dot{V}O_2_{max} = 55 \pm 2 \text{mL} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$) but a significant decrease of the constant time of the phase 2 ($\tau_2$) in the lowest-fit subjects ($\dot{V}O_2_{max} = 40 \pm 1 \text{mL} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$). In the present study, the seven subjects with the lowest $\dot{V}O_2_{max}$ ($= 64 \pm 1 \text{mL} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$) had a decrease of $\tau_2$ from 21 to 14 s that did not reach significance ($P = 0.16$).

Similarly, no changes were observed during the training period in either E or ES group in the amplitude of the slow component. These results seem in contrast with previous studies (5,6,10,28) which showed that 6–8 wk of endurance training led to a significant reduction of the amplitude of the slow component in healthy subjects. However, in several studies (6,28), exercise intensity was not recalculated, and because LT and $\dot{V}O_2_{max}$ may have been improved after training, the relative posttraining intensity may have changed, even below the recalculated $\dot{V}O_2_{max}$ (6). Because exercise intensity is one of the main factors influencing the amplitude of $\dot{V}O_2$, the results of these studies are difficult to interpret. Moreover, Carter et al. (5) showed a significant decrease of the amplitude of the slow component at the same absolute pre- and post-training velocities but not when the relative “new” postraining intensity was used, which is in line with the present results. Indeed, in the two groups pooled, the increase of the absolute $V_{\Delta25\%}$ ($P < 0.01$) was not accompanied by an increase of the amplitude of the slow component. Gaesser and Poole (10) suggested that endurance training brings a change in the motor unit recruitment pattern, with less fast-twitch fibers recruited after training. Due to the 18% lower ADP/O ratio in Type IIb mitochondria, when compared with Type I (27), it would lead to a decrease of the amplitude of the slow component after training. Moreover, a correlation between the EMG activity (i.e., mean power frequency) and the rise of the $\dot{V}O_2$ during the slow component phase was reported (3), suggesting that the fast-twitch motor units are progressively recruited to offset the decreased power output caused by the fatigued motor units. It is of interest to note that the major part of the $\dot{V}O_2$ kinetics changes, if not all, occurred very early, in the first 2 wk, and that no further change were observed in the latest part of the training period (28).

To understand whether the type of training could cause muscular adaptations leading to a change of $\dot{V}O_2$ kinetics, further investigations are required with aerobically paired athletes performing more distinct types of training (i.e., heavy-strength-only vs endurance-only) training, which could lead to more pronounced biochemical and histological adaptations in the trained muscles.

In conclusion, the addition of HWT to the endurance training of well-trained triathletes was associated with significant increase in running performance (i.e., $V_{\dot{V}O_2_{max}}$) and an enhancement of running economy, probably determined by an improvement in lower-limb stiffness regulation, as a result of the concurrent strength and endurance training. This hypothesis must be tested more directly in further studies. An alternative mechanism could lie in the increase of the maximal lower-limb power. In contrast with previous studies conducted with recreational athletes, neither the endurance nor the endurance + strength training changed the $\dot{V}O_2$ kinetics between pre- and post-training during heavy exercise, where the relative intensities were kept identical. Because additional HWT led to improve maximal strength and running economy with no significant effects on the $\dot{V}O_2$ kinetics pattern in heavy constant-rate exercise, its inclusion in the training program of well-trained endurance athletes is recommended.

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