CONSEQUENCES OF OPTIMAL PACING STRATEGIES FOR 400-, 800- AND 1500-M RACES ON VO2 RESPONSE

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ABSTRACT

This study aimed to compare the evolution of oxygen uptake ($\dot{V}O_2$) in specifically trained runners during running tests based on the 400, 800 and 1500 m pacing strategies used by the elite runners intending to realize their best performance. The final velocity decreases significantly in the three distances, the slowest velocity in the last 100 m expressed relatively to the peak velocity being observed on 400 m (77 %) compared to 800 m (88 %) and 1500 m (96 %). Relative to the previously determined $\dot{V}O_{2\,max}$ values, the respective $\dot{V}O_{2\,peak}$ corresponded to 94 % (400 m) and 100% (800 and 1500 m). In the last 100 m, a decrease in $\dot{V}O_2$ was observed in all the participants for 400 (15.6 $\pm$ 6.5 %) and 800-m races (9.9 $\pm$ 6.3%), whereas a non-systematic decrease (3.6 $\pm$ 7.6%) was noted on the 1500 m. The amplitude of this decrease is correlated to the tidal volume decrease also recorded during the last 100 m of each distance ($r = 0.85$, $P<0.0001$) and to the blood maximal [lactate] values measured post 400, 800 and 1500 m ($r= 0.55$, $P<0.005$). The present data demonstrate that 800 m is close to the 400 m relatively to decreases in velocity and $\dot{V}O_2$.

INTRODUCTION

Pacing strategy, defined as the variation of speed for the duration of the race by regulating the rate of energy expenditure (Foster, Schrager & Snyder, 1994) is proposed to be a marker of underlying physiological regulation (Tucker & Noakes, 2009). Performances of gold, silver and bronze medallists when directly compared are within 1% of each other and even small variations in pacing strategy may have substantial effects on the race outcome (de Koning, Bobbert & Foster, 1999). Among the competitive athletics distances, only 400-, 800- and 1500-m races are expected to require substantially both aerobic and anaerobic systems (Hill, 1999), due to high relative values of oxygen uptake ($\dot{V}O_2$) (Hanon, Lepretre, Bishop & Thomas, 2010; Hanon, Leveque, Thomas & Vivier, 2008; Thomas, Hanon, Perrey, Le Chevalier, Couturier et al., 2005) and blood lactate concentration greater than 15 $mmol.l^{-1}$ that have been previously observed (Hill, 1999; Lacour, Bouvat & Barthélémy, 1990). So, an analysis of the optimal pacing strategies during these particular athletic competitions potentially can inform coaches and researchers, and provide insight into the underlying physiological processes involved in the performance.

Few studies have focused on the pacing used in relevant athletics competitions. Tucker et al. (Tucker, Lambert & Noakes, 2006), who studied the pacing strategies during world-record performances for 800, 5000 and 10000 m races pointed out key differences between 800 m where it was observed that the second lap was significantly slower than the first lap and 5000 and 10000 m where an end spurt was observed due to the maintenance of a reserve during the middle part of the race. For these authors, the 800 and 5000 m races from the energetic point of view appear differently regulated. Hanon and Gajer (Hanon & Gajer, 2009b) described the pacing used by 400-m elite runners, and demonstrated that on this distance, the last 100 m was also the slowest of the entire race, as was the last 100 m of the 800-m elite races (Gajer, Hanon, Marajo & Vollmer, 2001).

To understand the reasons for the particular differences in race strategy indicates that there may be an impact on physiological responses. Manipulating start pacing strategies during the first 4 min of a 20

km cycling time trial, Mattern et al. (Mattern, Kenefick, Kertzer & Quinn, 2001) observed an impact both on the initial and subsequent $\dot{V}O_2$. The positive influence of an all-out pacing compared to constant pacing on a 2-min paddling performance has been demonstrated (Bishop, Bonetti & Dawson, 2002), explained by the authors as the result of a greater total $\dot{V}O_2$ with a similar accumulated oxygen deficit. Nevertheless, although demonstrated as optimal, these fast-start pacing strategies imply a velocity slow down (Gajer et al., 2001; Hanon & Gajer, 2009a) and a $\dot{V}O_2$ decrease (Billat, Hamard, Koralsztein & Morton, 2009; Hanon et al., 2010; Hanon et al., 2008; Thomas et al., 2005) at the end of the races. To date, no study has been conducted to compare the particular $\dot{V}O_2$ responses of these particular distances.

The present study aims to compare the actual pacing strategy adopted during athletics events when runners realized performances equal or very near their best performance, intending to describe and compare during experimental designs based on these strategies, the consequences on oxygen uptake. In light of past studies, since aggressive pacing strategy is necessary to obtain the best performance, and because of the supposed influence on metabolic responses, we hypothesize that 400, 800 and 1500 m could be characterized by gradual $\dot{V}O_2$ responses: i) a greater and earlier $\dot{V}O_{2\,peak}$ when the race distance is shorter, and the start velocity greater; ii) a greater $\dot{V}O_2$ decrease when the race distance is short, and great the contribution of the anaerobic metabolism to the energetic needs. In order to address these hypotheses, this study revises and synthesises previous personal data already published by our team in a scientific or technical journal.

METHODS

Data of six studies of our team, performed between 2000 and 2008, were analysed. Part of the results of these studies were published as original articles in scientific publications (Hanon et al., 2010; Hanon et al., 2009a; Hanon et al., 2008; Hanon, Thomas, Leveque & Vivier, 2007; Thomas et al., 2005 or technical (Gajer et al., 2001; Hanon et al., 2007) and the goal was either the description of the optimal pacing strategies or the consequence of them on the oxygen uptake time course. The analysis of the pacing strategies were all presented for elite runners (comparative level of performance) aiming to realize a best timed performance (part A) although the ventilatory studies (part B) was realized with lower performing runners (interregional or national level) with the respect of these pacing strategies models on adapted chronometric bases. In order to homogenate the 400-, 800- and 1500-m data, new treatments have been applied that could have modified the previously published results. In particular, the same method of determination on the $\dot{V}O_2$ decrease (last 100-m values expressed relatively to the peak values) was applied to the three distances that induce a lesser $\dot{V}O_2$ decrease compared to the original 800-m study (24%). Furthermore, in the 1500-m study (Hanon et al 2008) the $\dot{V}O_2$ decrease was not presented. The three studies of the part B conformed to the recommendations of the Declaration of Helsinki, and participants gave voluntary written consent to participate in this experiment, which was approved by the local ethics committee.

PART A: CHRONOMETRIC VELOCITY PROFILE ANALYSIS

Velocity profiles of elite athletes competing at 400, 800 and 1500 m have been characterized with video analysis. Three groups of 20 to 50 elite athletes each were chosen for the 400, 800 and 1500 m. Of these groups were eliminated all the participants whom performances were 0.5 (400 m), 1 (800 m) and 1.5s (1500 m) below their best performance and all 800 or 1500-m runners who were not discernible on each of the successive distance marks. Finally, three groups of 5, 25, 10 athletes were analysed for the 400, 800 and 1500m. The averaged performances in each group corresponds to about

the same level of performance (44.4 s, 1min 43 s and 3 min 35 s) which are close to the best world performances for the athletics season considered in each study and that corresponds to 1220, 1232 and 1175 points, respectively (IAAF Scoring Tables 2008, Revised Edition).

Protocol

Velocities

Because of the race realized in lanes and the subsequent gaps between the corridors, the 400 m was analysed differently (additional cameras) in comparison to the two others distances (Hanon et al., 2009a) for more details). The 800 and 1500-m analyses were conducted from the images from personal video of national athletics coaches or from IAAF data. The times of passage were determined each 50 m (400) or 100 m (800, 1500) by means of a time code on the TV monitor. Sequences were digitized in order to register the very moment when each athlete passed the markers which had been filmed before. The average velocity for each segment was calculated from times recorded for the 50 or 100 m segments.

PART B: VENTILATORY PARAMETERS

Participants

During three different studies of our team (2005, 2008, 2010), 10, 6 and 12 specifically-trained 400, 800 and 1500-m runners (5 to 7 training sessions in a week) were asked to reproduce these models of pacing strategies. These athletes were successful in regional and national running races (average performance of 50.9 ± 1.2 s for the men and 57.4 $\pm$ 3.7 s for the two women, 1 min 55 ± 0.03 min and 3 min 56 ± 0.21 min in 400, 800 and 1500 m, respectively).

Protocol

For all the tests, oxygen uptake ($\dot{V}O_2$) were recorded continuously by means of a telemetric gas analyser (Cosmed K4, Roma, Italy) as previously described (Hanon et al.2010; Hanon et al., 2008; Thomas et al., 2005). Capillary blood sample was measured by the Lactate Pro analyser (Arkray,

Japan) (800 and 1500 m) and by a Biosen Lactate analyser (Biosen C-line analyser, EKF Industrie, Elektronik GmbH, Barleben, Germany) (400 m).

Incremental protocols

$\dot{V}O_{2\,max}$ and v-$\dot{V}O_{2\,max}$ (minimal speed at which the athlete was running when $\dot{V}O_{2\,max}$ occurred) were determined using an incremental test conducted on an outdoor track as previously described (Hanon et al 2008, 2010, Thomas et al, 2005).

400, 800 and 1500-m running tests

The warm-up was standardised according to a regular pre-event warm-up (15-20 min of jogging, stretching, sprints at the respective start velocity). Since 400, 800 and 1500-m distance performances were different between athletes, cardio-respiratory data obtained as a function of time (5 s central moving average) were averaged over 25-m distance interval (400, 800m) and 50-m distance interval (1500m). The highest 5 s -value recorded on each distance is considered as $\dot{V}O_{2\,peak}$. The end $\dot{V}O_2$ ($\dot{V}O_{2\,end}$) value was defined as the average of the last 100-m values and the decrease in $\dot{V}O_2$ ($\dot{V}O_2\,decrease$) was defined as the difference between the $\dot{V}O_{2\,peak}$. and the $\dot{V}O_{2\,end}$ expressed relatively to the peak values. Blood samples were taken from the ear lobe just before the tests and then at 1, 4 (400m), 3 and 5 (800 and 1500m) and 7 and 10 min (400, 800, 1500m) during a passive recovery. The start and final velocities corresponded to the mean velocity during the first and the last 100-m segments. The velocity measured at 25% was the average velocity during the 25-m (400, 800 m) or 50-m (1500 m) segment preceding 100, 200 and 350m after the onset of the test for 400, 800 and 1500 m, respectively.

Statistics

Data are reported as mean ± SD. Relationships between variables (running velocity at different times of the race, metabolic parameters and $\dot{V}O_2$ kinetics) were analyzed by a Pearson's correlation coefficient. The relationship between blood [lactate] (X) and the $\dot{V}O_2$ *decrease/increase (Y) was*

assessed by regression analysis. All statistical analyses were conducted using Statview software (version 5.0). The level of significance was set at $P < 0.05$.

RESULTS:

PART A

VELOCITY

Figure 1 about here

As seen in table 1, and in spite of the start inertia, the start velocities recorded during the three races are always higher than the respective mean velocity. During 400 and 800-m distances (fig 1), the peaks of velocity are reached early during the first (400 m) and second 100m (800 m) although the fastest 100m is covered between the 1200 and the 1300th-m of the 1500-m distance. The velocity in the last 100 m decreases significantly on 400-, 800- and 1500-m races and is the slowest of the distance on 400 and 800-m. Expressed relatively to the peak value, the slowest final 100m are observed on the shortest distances (table 1).

Table 1 Start and Final velocity

	400 m	**800 m**	**1500 m**
Mean velocity (m.s^{-1})	8.99	7.76	6.97
First 100 m (m.s^{-1})	9.06	7.86	7.35
Segment time (s)	11.04	12.73	13.61
Last 100 m (m.s^{-1})	8.12	7.32	7.13
Segment time (s)	12.31	13.65	14.02
Last 100 m (%)	76.60	88.40	95.94

The velocity is measured on the complete distance (mean velocity) and on the first and last 100-m of each distance (m.s^{-1}). The first and the last 100 m segment times in s correspond to the necessary times to cover the first and the last 100 m of each distance. The last 100 m expressed in % is expressed relatively to the fastest portion (50m for the 400m and 100m for the 800 and the 1500m) of each corresponding distance.

Insert figure 2 about here

Table 2 :

Peak values (Mean ± SD) measured during (cardiorespiratory variables) and after [lactate] the 400, 800 and 1500-m tests.

	$\dot{V}O_2$ peak ($ml.min^{-1}.kg^{-1}$)	$\dot{V}O_2$ peak (% $\dot{V}O_2$ ~~tri~~max)	Distance at $\dot{V}O_2$ peak (m)	$\dot{V}O_2$ decrease (% $\dot{V}O_2$ peak)	Peak [lactate] ($mmol.l^{-1}$)
400 m	54.2 ± 5.7	93.9 ± 3.9	192 ± 22	15.6 ± 6.5	22.0 ± 1.9
800 m	66.3 ± 2.3	103.3 ± 3.8	316 ± 75	9.9 ± 6.3	17.8 ± 1.4
1500 m	69.2 ± 6.5	105.1 ± 10.5	459 ± 59	3.6 ± 7.6	14.9 ± 0.9

$\dot{V}O_{2\ peak}$ was determined as the greater 5 s central moving average. $\dot{V}O_2$ *decrease* expressed relatively to the peak values was defined as the difference between the $\dot{V}O_{2\ peak}$.and the $\dot{V}O_{2\ end}$.

As observed in table and figure 2, $\dot{V}O_{2\ peak}$ was greater and reached later when the distance is long. A $\dot{V}O_2$ decrease all the more important when the distance is short was observed in all participants on 400 and 800-m races and in 9/12 participants on 1500-m. Furthermore, a decrease in the average tidal volume (V_T) has been systematically observed in the three distances. A decrease in VE (minute ventilation) has been observed in the 400 and 800-m and a decrease in RF (respiratory frequency) was noted in the only 400-m distance (figure 4). A high correlation between the $\dot{V}O_2$ and V_T values recorded during the last 100 m of each distance has been observed: $r = 0.85$, $P<0.0001$.

Figure 4 about here

RELATIONSHIPS BETWEEN PACING STRATEGY AND OXYGEN UPTAKE

The velocities measured at 25% of the distance (100, 200 and 350m after the onset of the test for 400, 800 and 1500 m, respectively) were the velocity data the most strongly correlated to the time to reach $\dot{V}O_2$ *peak*. The time to reach $\dot{V}O_2$ *peak* was shorter when the start velocity at 25% of the distance was high ($r = -0.79$, $P < 0.0001$). Other correlations (12,5 and 33% of the distance) with time to reach

$\dot{V}O_2$ *peak* were calculated and found to be less correlated (P < 0.0005 and P < 0.005, respectively). The amplitude of the $\dot{V}O_2$ *decrease* was related to the peak of [lactate] values measured post-400, -800 and -1500m (r = 0.55, P< 0.005, n= 28) but no relation was observed between velocity and $\dot{V}O_2$ *decrease* (r = 0.29, NS). In the 1500 m population, the $\dot{V}O_2$ *decrease* was correlated with the velocity at 25% of the distance (measured between 300 and 350 m) expressed in % v-$\dot{V}O_{2max}$ (r = -0.59, P < 0.05). The significant relation between the magnitude of the $\dot{V}O_2$ *decrease/increase* and the peak [lactate] values post-races (r= 0.55, *P*<0.005, r^2=0.30) indicates that 10 mmol.l^{-1} is the minimal value under which no $\dot{V}O_2$ *decrease* is likely to occur on such an exercise (figure 3) and that blood [lactate] could explain 30% of the magnitude of the $\dot{V}O_2$ *decrease.*

Insert Figure 3 about here

DISCUSSION

These distances, 400, 800 and 1500 m, are characterized by common (fast-start) but also distinct (final velocity) characteristics making each one a unique distance. From 400 to 1500 m, the links between pacing strategies and $\dot{V}O_2$ uptake are clearly demonstrated with two major results indicating: 1) a strong relationship between the velocities measured at 25% of each distance and the time to reach $\dot{V}O_2$ *peak*, and 2) the occurrence of a $\dot{V}O_2$ decrease which is related to the final peak [lactate] value.

CONSEQUENCE OF THE START VELOCITY

The pacing strategies used by the 400, 800 and 1500 m elite runners, present some common characteristics such as a fast start and a slow-down in the last 100 m (Gajer et al., 2001; Hanon et al., 2009a; Hanon et al., 2007). Nevertheless, contrary to the shortest distances during which no acceleration is observed in the second half of the race, the 1500-m peak velocity is observed 300 m before the finish line.

In contrast, with similar studies realized on a treadmill at the average constant velocity (Draper & Wood, 2005; Draper, Wood & Fallowfield, 2003; Spencer & Gastin, 2001), oxygen uptake has previously been shown to be maximally or near maximally requested (Duffield, Dawson & Goodman, 2005a, 2005b; Hanon et al., 2010; Hanon et al., 2008; Thomas et al., 2005) from 400 to 1500 m using this positive (athlete's speed gradually declines) pacing strategy. Some authors have tested the positive effect of this pacing on the amplitude of the $\dot{V}O_{2\ peak}$, which results in a better performance in 2-min paddling (Bishop et al., 2002), running (Gastin, Costill, Lawson, Krzeminski & McConell, 1995; Sandals, Wood, Draper & James, 2006) and 60-s cycling (de Koning et al., 1999) exercises. The correlation ($r = -0.79$, $P < 0.0001$) observed in the present study between the velocity at 25% of each distance and the time to reach the $\dot{V}O_{2\ peak}$ ($r = -0.79$, P 0.0001) is in agreement with the literature (Astrand & Saltin, 1961; Hill, 1999). Therefore, this positive or quite all-out pacing strategy appears necessary to reach a high relative oxygen uptake in such a short time, and to obtain the best performance.

$\dot{V}O_2$ DECREASE IN THE LAST PART OF THE RUNNING DISTANCES

In our three distinct studies, a decrease in $\dot{V}O_2$ had been observed in the final part of the race while the velocity was always greater than the v-$\dot{V}O_{2\,max}$ for all subjects even in the last 100 m of the 400, 800 and 1500-m distances. Whereas this phenomenon was observed in every subject (400 and 800 m), this decrease was less important and not systematic during the 1500 m (Billat et al., 2009; Hanon et al., 2007). This $\dot{V}O_2$ *decrease* has been shown to be related to the final significant velocity decrease in 400- and 800-m distances but not in the 1500-m study, no more than observed in the total population (400, 800 and 1500 m). Nevertheless, for 1500 m, this final $\dot{V}O_2$ response was correlated with the velocity at 25% (300-350 m) expressed in % v-$\dot{V}O_{2\,max}$ indicating that the consequence of the pacing regulation depends on the exercise duration, making the 1500-m race differently regulated than the 400- and 800-m distances. Furthermore, as a $\dot{V}O_2$ *decrease* has previously been mentioned (Nummela & Rusko, 1995) and studied (Perrey, Candau, Millet, Borrani & Rouillon, 2002) at a constant pace on exhaustive treadmill running tests, the relationship between decreases in velocity and $\dot{V}O_2$ cannot be estimated as an evident causality relationship. During treadmill running tests to exhaustion, only biomechanical adaptation such as a decrease in stride length is possible (Hanon, Thepaut-Mathieu & Vandewalle, 2005). This decrease in stride length could be the result of a progressive failure to produce force (Taylor, Bronks, Smith & Humphries, 1997), since (Nummela, Vuorimaa & Rusko, 1992) found that drop-jump performance was impaired by approximately 39% following a maximal 400-m sprint, during which a stride length decrease was observed. The significant correlation between the magnitude of the $\dot{V}O_2$ *decrease* and the blood lactate concentration observed in the present study, suggests that this phenomenon is related to the high implication of the glycolytic contribution, and therefore to a large level of fatigue as shown by the decrease in stride length and/or stride frequency. To reinforce this result, a correlation was observed between final pH values and the final $\dot{V}O_2$ on the 400-m race (Hanon et al., 2010) . According to the present results and confirmed by the literature (Billat et al., 2009; Nummela et al., 1995), a large implication of the glycolytic contribution could

partly explain why a $\dot{V}O_2$ decrease is likely to occur during short and intense exercise. This relation between the magnitude of the $\dot{V}O_2$ *decrease* and the blood [lactate] could explain the absence of a $\dot{V}O_2$ decrease in the comparative results obtained with a less aggressive pacing regulation (Duffield, Dawson & Goodman, 2004; Duffield et al., 2005b; James, Sandals, Draper, Maldonado-Martin & Wood, 2007). One can then postulate that $\dot{V}O_2$ and velocity (or only stride length) decreases are both the consequences of an extreme fatigue.

Furthermore, the strong positive relationship observed between V_T and $\dot{V}O_2$ recorded during the last 100 m of the three races could be a consequence of the hyperpnea due to the metabolic acidosis (Forster & Pan, 1995). These results could indicate that the runners hyperventilated in order to partially compensate for their metabolic acidosis and to maintain an effective alveolar O_2 pressure (Miyachi & Katayama, 1999). This decrease in V_T associated with an increase in RF, observed in the 800 and 1500-m distances have been previously observed by (Cibella et al. 1996) (Cibella, Cuttitta, Kayser, Narici, Romano et al., 1996), during exhaustive exercise under chronic hypoxia and by Perrey et al (2002) (Perrey et al., 2002) in the subjects who demonstrated a $\dot{V}O_2$ decrease. Gallagher et al. (1985) (Gallagher, Im, Hof & Younes, 1985) have shown that this particular respiratory pattern observed in exhausting exercise could be considered as an indirect sign of respiratory fatigue. Nevertheless, it is to note that a decrease in all ventilatory parameters (VE, RF, V_T) was observed in the 400-m runners, demonstrating another respiratory pattern.

WHY IS THIS PACING STRATEGY THE MOST~~RE~~ EFFICIENT?

This pacing regulation also possesses the potential to cause premature fatigue because of disturbances in muscle pH. This acidosis has been shown to inhibit aerobic capacity (Jubrias, Crowther, Shankland, Gronka & Conley, 2003) and to cause low final $\dot{V}O_2$ values (Hanon et al., 2010; Thomas et al., 2005). As expressed by Foster et al. (Foster et al., 1994), athletes learn how to sense low values of muscle pH and adjust their pace accordingly, so that they ideally reach critical low values of pH near the end of a race. Interestingly, in 1997, Kipketer managed to beat the world record on 800 m only when he tried to use this pacing strategy. Interestingly, the pacing strategies described in this study confirm the model

based on the two equations derived from the Newton's second law and that for power balance of metabolism described by Maronski (1996). In this model, the author concluded that the optimal tactic to generate the minimal time on these intermediate distances, that are not sprint none long distances, implies an initial acceleration phase and a final decreasing velocity.

Based on the intermediate pH values recorded on 400 m (Hanon et al., 2010), well-trained athletes appear to be able to run about 15 s with a pH of 7.10, and must break off the pace when the pH values fall under 7.0. Many authors to date (Abbiss & Laursen, 2008; Billat et al., 2009; de Koning et al., 1999; Foster et al., 1994; Hettinga, De Koning, Broersen, Van Geffen & Foster, 2006) have concluded that anaerobic resources seem to be critical in regulating pacing strategy. Successful performance on 400 and 800 m must be realized with a full use of the buffering capacity (Ward-Smith, 1999), and therefore critical final values of pH (Foster et al., 1994). To accelerate in the last straight line is not compatible with these criteria, and does not allow for producing the best performance.

The 1500-m distance appears to be differently regulated compared to the shortest distances as there was a significant and negative relationship between start velocity, expressed in expressed in % v-$\dot{V}O_{2max}$) and final performance. This result is in good agreement with the conclusions of Billat et al (2009) who demonstrated that the 1500-m runners who had the longest limits at their anaerobic power in the first part of the race (that is closed to the present notion of velocity expressed in % v-$\dot{V}O_{2max}$) were those who achieved the best final performance. As in the Billat's study, this relation was obtained only in the 1500-m distance that indicates a different pacing regulation compared to the shortest distances. Furthermore, the difference in the level of the subjects (more than 20 sec between the present study and that of Billat et al (2009)) could indicate that this result depends on the exercise duration rather than on the level of training.

CONCLUSION: When exercise is less than 3 min 30 s, the only way to ensure the total depletion of the anaerobic reserves is to reach the exact sustainable pH value at the finish line. It appears that it could be done only with a fast start, and a slow-down in the last meters of the races as calculated by Maronski (Maronski, 1996). The decrease in $\dot{V}O_2$ observed in the final part of the three distances

examined herein appears as a specific consequence of aggressive pacing strategies and therefore, to an extreme exhaustion. The high correlation between the decreases in $\dot{V}O_2$ and V_T indicate that the ventilatory regulation likely induced by metabolic H+ accumulation is the major cause of the $\dot{V}O_2$ decrease.

The 1500-m race, differently regulated compared to the shortest distances, appears as intermediate between predominantly anaerobic distance (400 and 800 m races) and predominantly aerobic middle and long distance running.

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FIGURES

Figure 1. Time course of velocity during the 400-, 800-, 1500-m running elite competitions. Velocities expressed relatively to the peak of velocity of each distance (white, grey and black symbols correspond to 400, 800 and 1500-m velocities and respective arrow correspond to 400, 800 and 1500-m peak velocities). The 400, 800 and 1500-m velocities are presented every 25, 50 and 100 m, respectively.

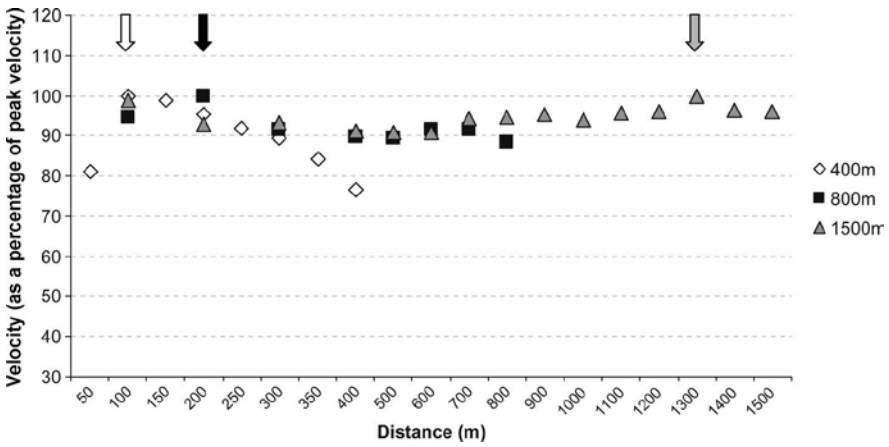

Figure 2. Time course of _V O2 during the 400-m (◊), 800-m (■), and 1500-m (▲) running tests.

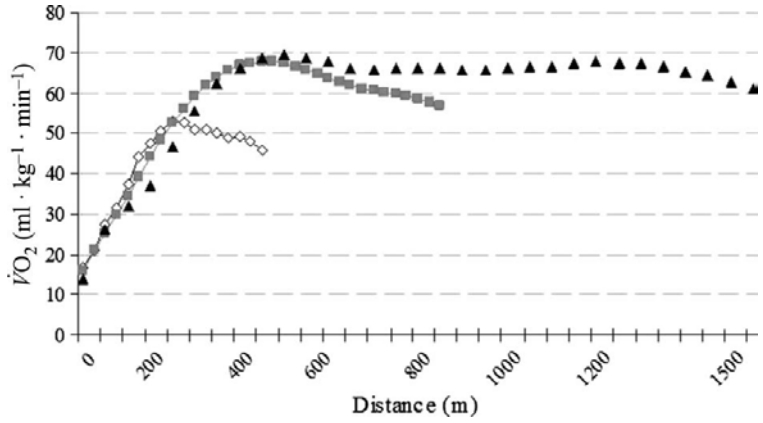

Figure 3. Ventilatory parameters time course during 400, 800 and 1500-m running tests. RF: respiratory frequency, VT: tidal volume, VE: minute ventilation measured during the 400-m (◊), 800-m (■) and 1500-m (▲) distances.

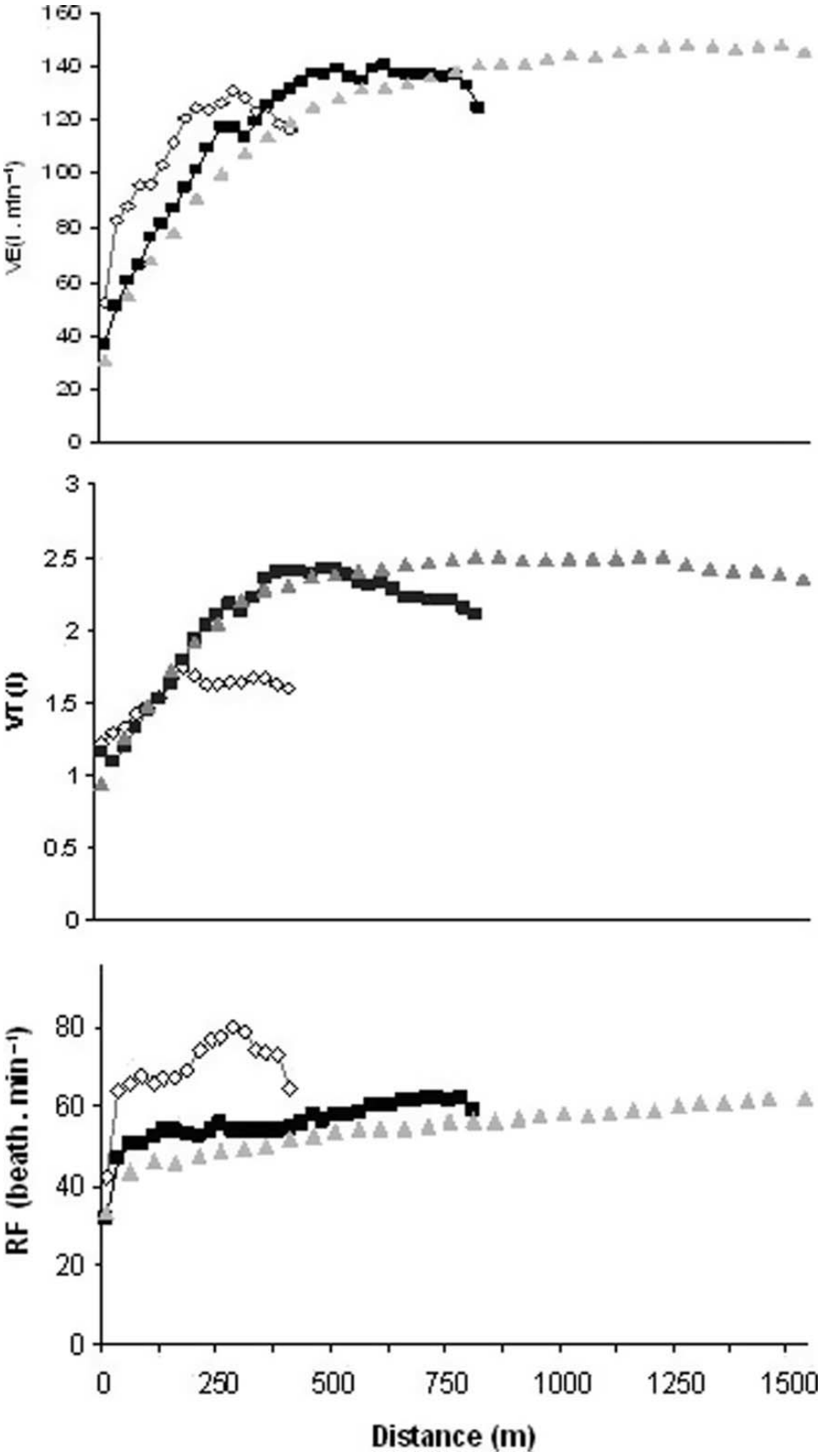

Figure 4. Blood lactate concentration (mmol . L^{-1}) vs. change in VO2 (ml . kg^{-1} . min^{-1}).